placental and neonatal blood cultures that this patient had an intrauterine infection with *Shigella* sp. To our knowledge, this is the first case to better document *Shigella* infection and its association with chorioamnionitis leading to preterm PROM and preterm delivery, as well as the first case of congenital shigellosis.

Armor et al⁴ reported a case of *Shigella* chorioamnionitis in a postterm patient resulting in neonatal *Shigella* bacteremia and pneumonia. Ruderman et al⁵ reported a preterm delivery at 30 weeks in which postpartum maternal stool culture revealed *S sonnei*, whereas an asymptomatic neonate had *S sonnei*-positive blood cultures. However, in this report, placental, amniotic fluid, or vaginal maternal cultures were not described. Therefore, causal association for the preterm delivery in their case report can only be inferred. In fact, the neonatal bacteremia may have been secondary to maternal transmission in the postnatal period.

Most intrauterine pathogens in pregnancy are believed to ascend the vaginal vault and cervix and cause infection as they travel along the chorioamniotic membranes. In our patient, both maternal and fetal membrane cultures were positive for *Shigella* at time of delivery. The upper genital tract cultures were positive for *Shigella*, whereas the lower vaginal tract cultures revealed normal vaginal flora. We speculate that in this case intrauterine infection was not attributable to vaginal colonization, but to maternal hematogenous spread. *Shigella* is a highly virulent enteroinvasive organism, which has been reported to result in bacteremia/sepsis.

Interestingly, this preterm fetus of 26% weeks passed meconium before delivery. Meconium is seldom passed before 34 weeks. Its presence is often believed to reflect gastrointestinal maturity in late gestation, as the immaturity of intrinsic and extrinsic innervation of the bowel may impair the ability of the premature fetus to pass meconium. Furthermore, this fetus showed little hypoxic insult as revealed by a reassuring fetal heart tracing and

cord gases at time of delivery. We must, therefore, question the reason for passage of meconium in an "unstressed" preterm fetus. Perhaps the passage of meconium is related to the fetal swallowing of *Shigella*-infected amniotic fluid resulting in fetal shigellosis. The *Shigella* toxin damages the mucosal layer of the colon, thus limiting absorption of fluids and electrolytes and resulting in a liquid stool. Positive fetal stool cultures and negative fetal blood cultures at delivery indicate that the infection may have been localized to the fetal/neonatal gastrointestinal tract.

As preterm PROM and its associated preterm delivery have significant morbidity and mortality, it is prudent to consider *Shigella* infection in symptomatic patients. Clinicians should consider treatment of symptomatic patients (ie, bloody diarrhea) empirically, particularly those patients with risk factors for *Shigella* infection. Neonatal testing is indicated if maternal disease is suspected before delivery.

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Dysmenorrhea After Bilateral Tubal Ligation: A Case of Retrograde Menstruation

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Pediatric and Reproductive Endocrinology Branch, National Institute of Child Health and Human Development, Bethesda, Maryland; and Georgetown University Medical Center, Washington, District of Columbia BACKGROUND: Endometriosis, arising de novo, is believed to be uncommon in women who have undergone bilateral tubal ligation because the occluded tube prevents outflow of blood and menses.

CASE: A woman 10-year status-post bilateral tubal ligation suffered from dysmenorrhea and menorrhagia that began within 1 year after sterilization. At the time of bilateral

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tubal ligation, no endometriosis was observed. A recent magnetic resonance imaging scan showed no pelvic abnormalities, and the patient underwent a diagnostic laparoscopy in anticipation of finding endometriosis, yet none was found. At laparoscopy performed on day 3 of her menstrual cycle, the proximal segments of her occluded fallopian tubes were dilated with blood. As this was the only abnormality found, we postulated that her dysmenorrhea might be related to the dilated proximal tubal stumps. We evacuated the bloody fluid and occluded the proximal tube at the cornua with Filshie clips. One year after surgery, the patient remains asymptomatic.

CONCLUSION: This case is unique because bilateral tubal ligation combined with retrograde menstrual flow appears to have caused dysmenorrhea. Women who have undergone tubal ligation and who have dysmenorrhea may benefit from a diagnostic laparoscopy during menstruation to evaluate the possibility of retrograde menstruation dilating the proximal tubal stumps. (Obstet Gynecol 2002; 100:1065–7. © 2002 by The American College of Obstetricians and Gynecologists.)

Bilateral tubal ligation is one of the most prevalent and effective contraceptive methods, with a 0.4% pregnancy rate after 1 year and a cumulative failure rate of 1.85% after 10 years.^{1–3}

Endometriosis, a disease in which endometrial glands and stroma grow outside the uterus, may develop after retrograde menstruation and seeding of the peritoneal cavity with endometrial cells. The development of endometriosis is uncommon in women who have undergone bilateral tubal ligation because the occluded tubes do not allow the outflow of menstrual fluid into the peritoneal cavity. In the present case, we describe a woman who had dysmenorrhea after bilateral tubal ligation, presumably from proximal tubal segments dilated with blood as a result of retrograde menstruation.

CASE

A woman 10-year status-post bilateral tubal ligation presented with a 10-year history of worsening dysmenorrhea and menorrhagia. At the time of bilateral tubal ligation, no endometriosis or other pelvic pathology was noted. The patient tried taking oral contraceptive pills to alleviate the pain without relief. Pelvic examination was unremarkable. A recent pelvic magnetic resonance imaging scan was normal with a mildly prominent endometrial cavity, small follicles in the right ovary, and a 1.6-cm cyst in the left ovary. The cyst was believed to be a normal physiologic follicle. Endometriosis was suspected, and she enrolled in a study of the clinical management of endometriosis. At laparoscopy performed on day 3 of her menstrual cycle, there was no evidence of

endometriosis. However, the proximal portions of both ligated fallopian tubes were dilated with menstrual blood. Bipolar cautery scissors were used to open the distal ends of the proximal tubal segments and drain the bloody fluid. Filshie clips were placed at the cornuas of the proximal portions of both tubes. The abdomen was suction lavaged, and the tubes were then recauterized. No postoperative complications occurred. During the year since surgery, she no longer has dysmenorrhea.

COMMENT

Women who have undergone tubal ligation are no more likely than other women to have menstrual abnormalities, and most patients experience no long-term complications after bilateral tubal ligation. Leaving a 2- to 3-cm stump minimizes the risk of fistula formation, allows for tubal reversal, and reduces postsurgical complications.

However, in our patient, the proximal stumps filled with menstrual fluid during menstruation. Previous reports have suggested that retrograde menstruation occurs in 90% of all women,⁷ although it is not generally associated with pain, presumably because the menses flow into the abdominal cavity rather than dilating the fallopian tube. In this case, because drainage of the fluid and occlusion of the cornual junctions improved pain immediately, we infer that tubal dilatation from the collection of blood was the cause of her severe menstrual pain. A previous study investigating the cause of pelvic pain in women who had tubal ligation followed later by endometrial ablation found that the proximal portions of one or both tubes were dilated in every case.⁸

These observations suggest that for women who complain of dysmenorrhea after bilateral tubal ligation, laparoscopy should be performed during menstruation to determine whether retrograde menstrual flow is causing dilatation of the proximal fallopian tubal segments. Had this operation not been performed during menstruation, dilatation of her occluded tubes may not have been discovered. Thus, women who have undergone tubal occlusion and who have dysmenorrhea may benefit from a diagnostic laparoscopy.

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Pain in the Foot: Calcaneal Metastasis as the Presenting Feature of Endometrial Cancer

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BACKGROUND: Ninety percent of endometrial cancer cases present with abnormal bleeding. Bone metastasis as the presenting feature is extremely rare.

CASE: A 76-year-old woman presented with right heel pain. She had no vaginal bleeding or other symptoms suggestive of endometrial cancer. After failure of conservative therapy, imaging studies demonstrated a calcaneal metastasis. A biopsy showed adenocarcinoma. She received local radiation to her foot, with complete resolution of symptoms. Subsequent computed tomography scans showed multiple pulmonary nodules, pelvic and inguinal lymphadenopathy, and an enlarged uterus. Endometrial biopsy confirmed endometrial adenocarcinoma. She received palliative therapy and died 11 months after the diagnosis was made on the endometrial biopsy.

CONCLUSION: This case highlights the rare presentation of endometrial cancer with foot pain secondary to calcaneal metastasis. Aggressive treatment of bone metastases can provide effective palliation of symptoms. (Obstet Gynecol 2002;100:1067-9. © 2002 by The American College of Obstetricians and Gynecologists.)

Endometrial carcinoma is the most common gynecologic malignancy, with over 36,000 cases occurring annually

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in the United States.¹ Abnormal vaginal bleeding is the presenting symptom in 90% of cases, and 73% are early stage with disease apparently confined to the uterus.¹ Bone metastasis, occurring in 5–6% of cases,² is a very rare cause of initial symptoms. We report a case of endometrial adenocarcinoma presenting with isolated foot pain from a calcaneal metastasis.

CASE

A 76-year-old woman presented with right heel pain, which began in September 1999. She was initially treated for a presumed diagnosis of plantar fasciitis without symptomatic improvement. Subsequent x-rays of the right foot revealed a large, lytic lesion of the right calcaneus (Figure 1), and bone scintigraphy confirmed this to be a likely isolated metastasis (Figure 2). A computed tomography-guided needle biopsy confirmed metastatic adenocarcinoma of unknown primary. A diagnostic workup was initiated, and she commenced external beam radiation to the right heel, which was successful in alleviating her pain. Subsequent computed tomography scans revealed pulmonary nodules, an enlarged uterus,



Figure 1. Lateral x-ray of the right foot demonstrating lytic lesion of the calcaneus.

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